Case Record 2

Branch Vein Occlusion.

June 2012

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aaron optometrists

The need for 'Desk Side' reference sources to ensure evidence based management and self reflection.

The current Royal College of Ophthalmologists guidelines on the management of retinal vein occlusion (RCO 2010) are designated as 'interim', reflecting the rapidity in development of treatment options. For the ever increasing number of clinical optometrists, wishing to become valuable members of clinical management networks, ready access to current literature is essential. Optometrists, often the initial diagnosticians, should initiate concurrent medical management and advise both GP and patient. Detecting underlying medical conditions ensures prompt management of systemic disorders as well as preventing recurrences of BRVO, potentially in the contraleteral eye (RCO 2010).

However there is a lack of consensus in the literature on pathogenesis, natural history, risks of macular oedema and neovascularisation and visual prognosis (McIntosh et al 2007, Hayreh 2005). Often grouped as sub-categories of retinal vein occlusions (RVO), branch and central vein occlusions represent fundamentally different entities (Wu 2012, Hayreh 2005). Wu (2012) suggests that most cases of branch retinal vein occlusions (BRVO) are idiopathic. BRVO typically occur at arteriovenous crossings where the artery and vein share a common adventitial sheath (NICE 2009); artery wall hardening due to aging and systemic hypertension cause obstruction of venous blood flow at this crossing (Christoffersen et al 2007, Klein et al 2008). Resultant loss of lamina blood flow causes secondary thrombosis, macular oedema and decreased visual acuity (Cheung et al 2008). Historically many medical treatments have been espoused for Branch Retinal Vein Occlusion (BRVO), including anticoagulants, fibrinolytic agents, cholesterol reducing agents and haemodilution to reduce plasma viscosity (Wu 2012). Hayreh (2007) insists that these, often continued, misconceptions are the result of not considering BRVO as an independent morbidity with quite specific pathophysiology. Certainly Cheung et al (2008) suggest that the primary risk factors for BRVO is systemic hypertension, no evidence of systemic inflammation, endothelial dysfunction, coagulation diseases or artherosclerosis of carotid, coronary or peripheral circulation could be presented. Further, in the five year longitudinal Beaver Dam Study, Klein et al (1999) stated that retinal emboli did not develop in any eye with ateriovenous nicking at baseline.

Busy practitioners are unlikely to critically appraise research; availability of desk side evidence based and peer reviewed e-resources are readily

available and constitute a vital tool to supply up-to-date advice to both patient and GP. Emedicines is the primary desk side resource; while registration is required, this resource is free to health care practitioners.

March 2011

Salient information taken from electronic records

DATE: 16/3/11

Mrs Age : 70.

Presenting Symptoms

2/52 history of migraine. Visual disturbance with headache. During migraines noticed scotoma in left eye but not noticed since.

POH

Reading Specs only. No previous ocular surgery or treatments.

FOH

Glaucoma.

General Health and Medications

Non-smoker. No allergies, No hayfever

No medications : General health good, but has not had health check for several years.

No previous history of general or ocular medication use or surgery.

Refraction

R +1.00/-0.75x100 (6/6) Add +2.25 N5

L +1.75/-0.75x85 (6/7.6-) Add +2.25 N5

Phorias Dist- 3Exo Near Orthophoric

Tensions (GAT) (11.05am) R 19 L 20

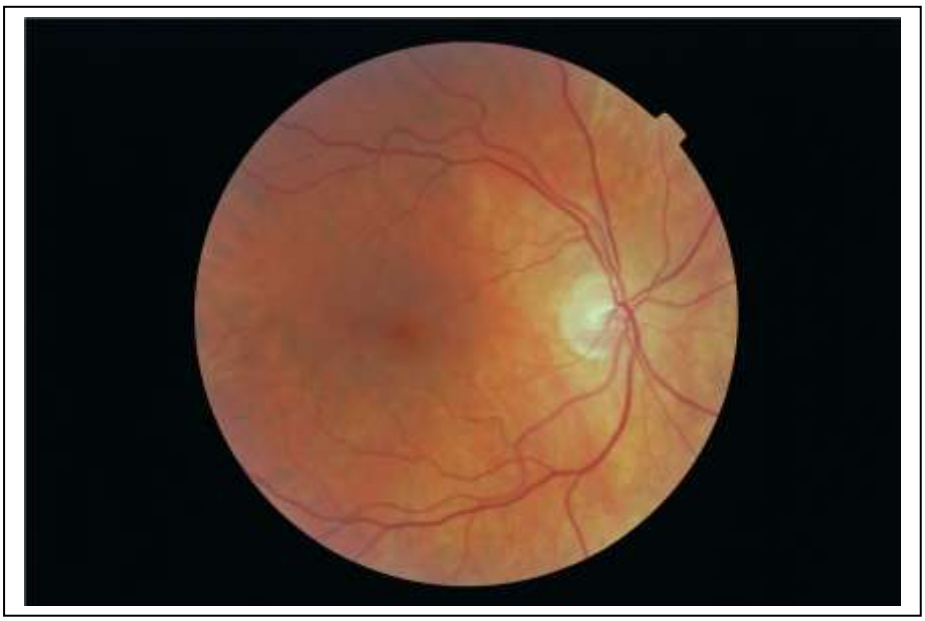
Pupils E&A D,C& N

Slit Lamp

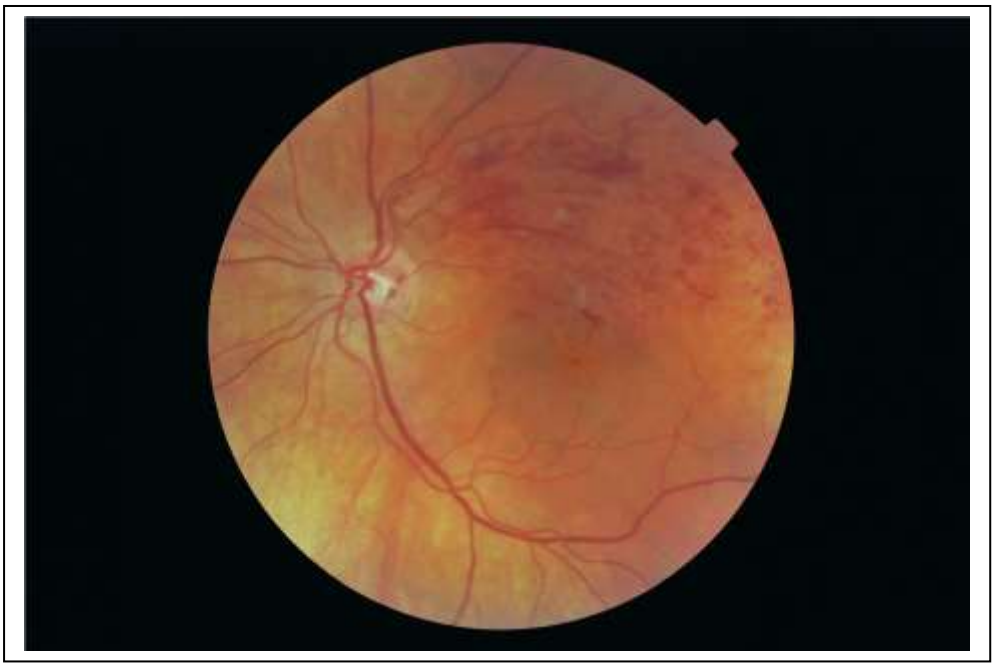
VH 3 Angles open, Iris configuration Flat. AC Clear – no pigment. Corneas clear.

Dilated Fundsocopy (1.0% Tropicamide)

RIGHT CD 0.2 rims good, no bayoneting, no baring. Neural rims healthy. AV 2/3, no nipping, no calibre changes.



LEFT CD 0.2; superior rim congestion and haemorrhages associated with superior branch vein occlusion immediately adjacent to disc, within superior arcade and impinging on macula with macula oedema.



DDx Hemicentral Retinal Vein Occlusion?

Advice and CMP

Advised Px with fundus photos. HES contacted due to macula involvement and advised to refer directly within 24/24. Report sent to GP suggesting blood screens for underlying co-morbidities.

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INDEPENDENT Prescribing
OPTOMETRISTS
Bellway House, Woodhorn Road, Ashington,
Northumberland NE63 0AE, Tel:01670 813185

Dr

Re

16/3/11

Dear Dr

Mrs presented reporting a two week history of inferior field loss in the LE.
Refraction gave:
R +1.00/-0.75x100 (6/6) Add +2.25 (N5)
L +1.75/-0.75x90 (6/7.6-) Add +2.25 (N6)

Dilated fundoscopy revealed a superior branch vein occlusion in the left eye.

The macula is involved and I have contacted ophthalmology and Mrs will present to eye casualty tomorrow morning.

I have recommended a blood screen to identify any underlying anomalies.

Yours faithfully

Peter Frampton

Outcome Audit 2011

Salient information taken from electronic records

DATE: December/11

The Newcastle upon Tyne Hospitals NHS
NHS Foundation Trust

DEPARTMENT OF OPHTHALMOLOGY

Consultant Ophthalmologist

Mr E Barnes
Mr M Birch
Mr A Browning
Mr M P Clarke (Head of Dept.)
Miss L Clarke
Mr D G Cottrell
Miss M Dayan
Miss A J Dickinson
Mr F Figueiredo
Mr P G Griffiths
Mr R Gupta
Miss ZK Johnson
Mr C Neoh
Mr R Pandit
Mrs N Ray-Chaudhuri
Mr A Shafiq
Mr K P Stannard
Mr N P Strong
Mr S J Talks

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Direct line: 0191 2820474
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RG/LD/91091911
NHS:4250114279

Date of clinic: 12/12/2011

(115)

Infirmary Drive MED Group
The Consulting Rooms
Alnwick
Northumberland
NE66 2NR

Dear Dr

Re:

Diagnosis: Left supero-temporal branch retinal vein occlusion with cystoid macular oedema.
Left eye previous 4 Avastin injections last injection September 2011.

Vision: Right 6/9 and left 6/24 improving to 6/12+2 with pinhole.

The patient thinks vision is stable. Today's examination reveals no signs of anterior or posterior segment vascularisation from the left eye. No signs of macular oedema on fundoscopy.

Plan: Observe.

Follow-up: 6 to 8 weeks.

If the patient notices visual deterioration we recommend attends Eye Casualty.

Yours sincerely

October 2011

Salient information taken from electronic records
DATE: 11/11/11

Mrs Age : 71.

Presenting Symptoms
Vision seems stable- blur is disappearing. No headaches, no diplopia.

POH
LE Superior Branch Vein Occlusion. Avastin Injections X4. Under HES review 3/12ly HES checks

FOH
Glaucoma.

General Health and Medications
Non-smoker. No allergies, No hayfever
Simvastatin
General health good otherwise – has not been put on Aspirin.

Refraction

R +1.25/-0.75x100 (6/6)	Add +2.25 N5
L +1.75/-0.75x90 (6/7.6-)	Add +2.25 N5

BRVO Treatment Options

Wu (2012) stated that medical management of BRVO is not effective. The original referral letter to the GP recommending blood screens will have been of little specific benefit for the BRVO. The patient is now on Simvastatin, but this will have been an intervention coincidental to BRVO management. Advice to try preventing a similar episode in the fellow eye does not seem to be valid considering the pathophysiology of BRVO. Christoffersen (2007) state systemic factors alone are not sufficient to create BRVO and BRVO does not appear to predict higher rates of stroke or mortality (Ho et al 2009, Christoffersen et al 2007). Regardless, congestion of the superior disc was noted and a clear site of AV occlusion was not evident. While the extent of retinal involvement would not suggest hemicentral vein occlusion (Hemi-CRVO) this possibility was considered as a differential. Hemi-CRVO is more akin to

central retinal vein occlusion (CRVO) and would be more likely to require management of systemic disorders (Kooragayala 2011).

A number of treatment modalities have been used to varying degrees of success; various laser techniques, intravitreal corticosteroids, hemodilution, surgical procedures of vitrectomy and adventitial sheathotomy, and AntiVEGF drugs (McIntosh 2007). Acute BRVO is often monitored as spontaneous resolution may occur within three months (McIntosh et al 2007). This recommendation could reflect the modest improvements and iatrogenic risks of some of the older treatments. Coupled with the very low risk of neovascularisation and neovascular glaucoma in BRVO (Wu 2012, Hayreh 2005) made monitoring an acceptable management option.

However, the Royal College of Ophthalmologist guidelines (2010) recommend either an intravitreal implant of Dexomethosone (Ozurdex) or Ranibizumab (Lucentis) intravitreal injections for patients with macula oedema secondary to BRVO if seen within three months.No suggestion of delay is mentioned. The college states Lucentis is not licensed for this use although the Summary of Product Characteristics (SPC) does specify the drug is indicated for vision loss due to macula oedema post both CRVO and BRVO (eMC 2012) but conclude there is currently insufficient evidence as to when ranibizumab treatment should be initiated.
The College of Ophthalmologists also give advice on the use of Bevacizumab (Avastin). Licensed for bowel, breast and small cell lung cancer (eMC 2012) it has been demonstrated to have more adverse reactions than Lucentis (CATT research group 2011) and the College of Ophthalmologists (2010) suggest that the GMC Good Medical Practice Guidelines and the manufacturer's advice should guide the intraocular use of Bevacizumab. Anti-VEGF drugs are revolutionising the treatment of many retinal disorders. Noma et al (2005) demonstrated that levels of both Vascular Endothelial Growth Factor (VEGF) and Interleukin-6 (IL-6) are elevated in the aqueous humour of eyes with BRVO. Both these chemicals appear to originate intra-ocularly and expression reflects levels of ischaemia. The authors also suggest that IL-6 indirectly induces VEGF expression suggesting that targeting VEGF rather than IL-6 would be most efficacious.

The ophthalmologist caring for this patient chose to use Avastin. The results have been excellent and no adverse reaction, systemic or ocular, has been reported. The responsibility of using an unlicensed drug would be difficult; weighing up risks, benefits and costs. Ocular adverse effects

listed in the summary of product characteristics (eMC 2012) are similar for both Lucentis and Avastin and may well reflect surgical technique rather than direct drug reaction. Inflammations (uveitis, vitritis) Haemorrhages (Retinal, Vitreal, Hypopyon), Retinal Detachments and Tears, Endophthalmitis and traumatic cataract are serious adverse reactions listed for Lucentis and intuitively the majority reflect general risks of intraocular procedures. Similar reactions are listed for the unauthorised intraocular use of Avastin although the frequencies are not mentioned.

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